

# Echocardiographic assessment of valvular pulmonary stenosis in children\*

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**SUMMARY** It has been suggested that the maximal amplitude of the pulmonary valve motion following atrial contraction (Amax) may be useful in the clinical evaluation of patients with valvular pulmonary stenosis. To evaluate the specificity and sensitivity of this measurement in children, we reviewed the echocardiograms of the pulmonary valve of 120 subjects: 57 normal individuals, 25 patients with secundum atrial septal defect and without pulmonary hypertension, and 32 patients with pulmonary stenosis proven at catheterisation (mild in 16 patients, moderate in nine, and severe in seven). Amax ranged from 0 to 12 mm in the normal subjects, and from 2 to 16 mm in those with atrial septal defect. In mild pulmonary stenosis, Amax ranged from 2 to 12 mm, in moderate pulmonary stenosis from 2 to 14 mm, and in severe pulmonary stenosis, from 3 to 12 mm. Though mean Amax was significantly larger in patients with moderate and severe pulmonary stenosis compared with normal subjects, there was much overlap between the two groups so that individual cases could not be identified correctly from this measurement. No significant difference was observed when comparing Amax values of patients with atrial septal defect and those of patients with pulmonary stenosis of various severity, nor were any observed between the pulmonary stenosis groups. These findings indicate that Amax is neither specific nor sensitive for the presence or severity of valvular pulmonary stenosis in children, and that it cannot be used to evaluate non-invasively the results of pulmonary valvotomy.

Because of the position of the pulmonary valve in the chest and its plane of motion relative to the ultrasonic beam, the echocardiogram usually records only one posterior leaflet.<sup>1</sup> The "a" wave is that posterior deflection of the pulmonary valve echo which reflects the effect of atrial contraction on the pulmonary valve. This is confirmed by its temporal relation to the P wave of the electrocardiogram and its disappearance during atrial fibrillation. The amplitude of the "a" wave varies with respiration, increasing during inspiration, and its maximal amplitude is referred to as "Amax". In a study involving mostly adults,<sup>2</sup> it has been suggested that in patients with valvular pulmonary stenosis the maximal posterior displacement of the pulmonary valve echo following atrial contraction was significantly increased above normal values, and that the severity of the stenosis could be

predicted from the echocardiogram. The present study was undertaken in order to evaluate the specificity and sensitivity of Amax in children with various degrees of valvular pulmonary stenosis.

## Subjects and methods

We reviewed the pulmonary valve echocardiographic tracings of 120 subjects, who were divided into three groups.

Group 1 consisted of 57 normal subjects aged 3 to 23 years (mean 7.3 years) who had an echocardiogram as part of the investigation of a functional heart murmur.

Group 2 contained 25 patients aged 1 to 26 years (mean 11.6 years) with secundum atrial septal defect documented by cardiac catheterisation (Qp/Qs: 1.5-8.0). In all of them, the right ventricular pressure was less than 45 mmHg and the angiographic appearance of the pulmonary valve was normal. Fifteen of these patients were restudied after surgical correction of their defect.

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Group 3 consisted of 38 patients aged 2 to 33 years (mean 11.2 years) with isolated valvular pulmonary stenosis. Pulmonary stenosis was diagnosed on the basis of a right ventricular to main pulmonary artery pressure gradient at valvular level plus the angiographic appearance of a domed valve. This group was further divided in three subgroups according to right ventricular pressure.<sup>3</sup> There were 16 patients with mild pulmonary stenosis (right ventricular pressure <50 mmHg), 10 patients with moderate pulmonary stenosis (right ventricular pressure 50 to 100 mmHg), and 12 patients with severe pulmonary stenosis (right ventricular pressure  $\geq 100$  mmHg). Satisfactory records could not be obtained from one patient with severe and five with severe pulmonary stenosis, leaving nine and seven patients in these two groups respectively. Eight patients with severe pulmonary stenosis were restudied one year after successful pulmonary valvotomy as documented by a right ventricular pressure of less than 50 mmHg at cardiac catheterisation. The electrocardiogram showed sinus rhythm and a normal PR interval in all cases.

The subjects' weights were as follows. Group 1: 4.5 to 72.7 kg (mean 26.3); group 2: 5.0 to 68.0 kg (mean 34.3); and group 3: 12 to 75 kg (mean 30.5).

#### ECHOCARDIOGRAPHY

Echocardiographic examinations were performed with an Ekoline 20A echograph combined with a Honeywell 1856 fiberoptic strip chart recorder. Either a 2.25 MHz or a 3.5 MHz transducer was used depending on the size of the patient. The pulmonary valve echoes were recorded using the method described by Gramiak *et al.*<sup>4</sup> The amplitude of the "a" wave was measured on several cardiac cycles during quiet respiration and Amax was defined as the maximal amplitude recorded (Fig. 1).<sup>2</sup> When point "b" was clearly identifiable, the amplitude of the "a" wave was measured from the position of the pulmonary valve echo at the end of the P wave on the simultaneously recorded electrocardiogram to its most posterior position before or at point "b". Presystolic opening was considered to be present when the maximal posterior displacement of the pulmonary valve echo occurred after atrial contraction before ventricular contraction, so that point "b" could not be identified. In those cases, Amax was measured as the distance between the pulmonary valve echo at the end of the "P" wave and its position at the peak of the R or R' wave. The maximal amplitude of the posterior aortic wall motion after atrial systole (ao wave) was measured according to the method described by Pocosky and Shah.<sup>5</sup>

#### PRESSURE MEASUREMENTS AND ANGIOGRAPHY

Cardiac catheterisation was performed under sedation

with a mixture of pethidine hydrochloride (1 mg/0.45 kg) and promethazine hydrochloride (0.25 to 0.5 mg/0.45 kg). Right ventricular and main pulmonary artery pressures were measured through fluid-filled catheters. The pulmonary valve was evaluated angiographically in the left lateral projection after right ventricular injection of 1 ml/kg of Renographin 76.

Statistical analysis used Student's *t* test to compare the mean values of Amax of the different groups under study. Linear regression analysis was used to correlate Amax with the subjects' age and weight for each group, as well as to correlate the Qp/Qs and Amax values in patients with secundum atrial septal defect.

## Results

### AMAX (Table)

#### Normal

In normal subjects, Amax ranged from 0 to 12 mm (mean 4.8 mm). Respiratory variation in the amplitude of the "a" wave was observed in all subjects, but in none did the pulmonary valve echo return to the baseline during quiet respiration (Fig. 1). Complete presystolic opening of the valve was observed in 13 out of 57 normal subjects. Correlation between Amax and subjects' age or weight was poor, with *r* values of 0.03 and  $(-0.04)$ , respectively.

#### Secundum atrial septal defect

In the patients with secundum atrial septal defect, Amax ranged from 2 to 16 mm (mean 6.6 mm). After operation Amax ranged from 0 to 10 mm (mean 4.8 mm). Mean Amax was significantly greater than normal preoperatively ( $p < 0.05$ ) but not postoperatively. Complete presystolic opening of the pulmonary valve was frequently observed in these patients (Fig. 2). The increased Amax values in patients with secundum atrial septal defect were not related to the magnitude of the pulmonary blood flow ( $r = 0.37$ ), nor to the patient's age ( $r = -0.10$ ) or weight ( $r = -0.12$ ).

Table Amax values in three groups of patients studied

Groups	No.	Amax (mm) (mean $\pm$ SEM)	Range (mm)
1: Normal	57	4.8 $\pm$ 0.4	0-12
2: Atrial	25	6.6 $\pm$ 0.7*	2-16
septal defect	15	4.8 $\pm$ 0.7	0-10
3: Pulmonary	16	5.4 $\pm$ 0.8	2-12
stenosis	9	6.9 $\pm$ 1.2*	2-14
Moderate	7	7.3 $\pm$ 1.0*	3-12
Severe preop.	8	6.3 $\pm$ 1.0	2-9
postop.			

\* $p < 0.05$  compared with normal subjects

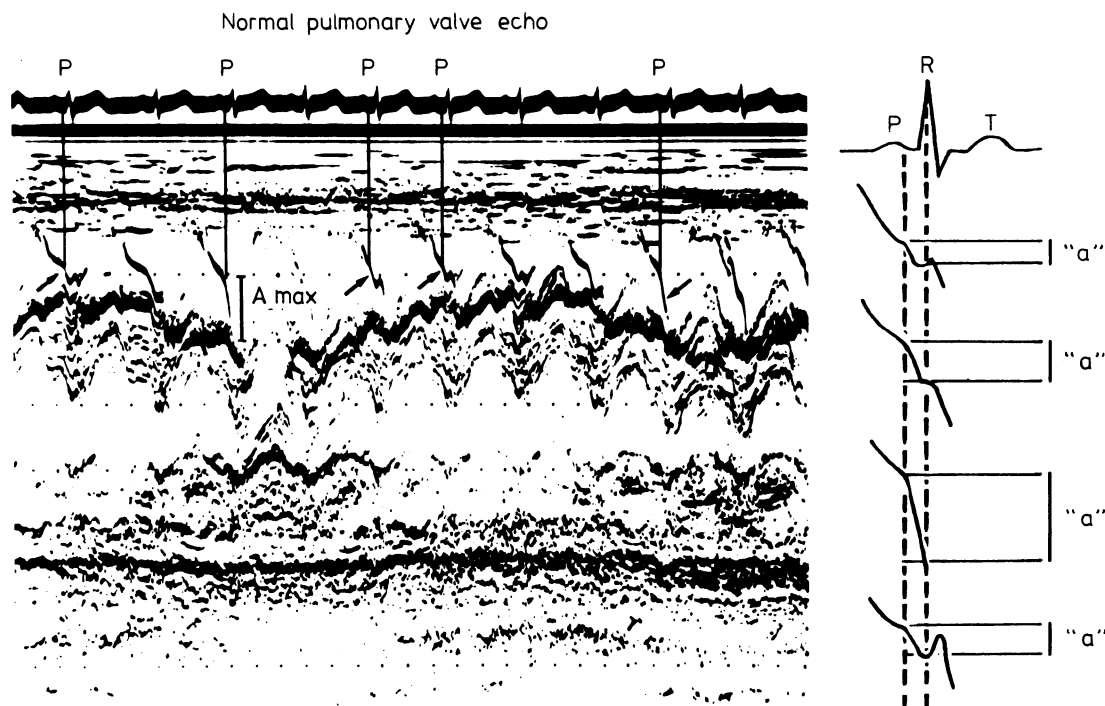


Fig. 1 Pulmonary valve echo (left) from a normal subject. The "a" wave follows atrial contraction and its amplitude varies (arrows). Amax represents its maximal amplitude. The 3rd and 9th cycle show presystolic opening of the valve (see text). Diagram (right) showing various morphologies of the "a" wave and how its amplitude is measured in each case.

#### Pulmonary stenosis

In the patients with mild pulmonary stenosis, Amax ranged from 2 to 12 mm (mean 5.4 mm). Patients with moderate pulmonary stenosis had an Amax range of 2 to 14 mm (mean 6.9 mm). In the seven patients with severe pulmonary stenosis Amax ranged from 3 to 12 mm (mean 7.3 mm). Mean Amax was significantly greater than normal in patients with moderate and severe pulmonary stenosis ( $p < 0.05$ ) but not in those with mild pulmonary stenosis. In the patients studied after successful pulmonary valvotomy, Amax ranged from 2 to 9 mm (mean 6.3 mm). This value was not significantly different from normal. Since the range of Amax values was the same in the three subgroups of patients with pulmonary stenosis, it was not possible to evaluate the severity of the stenosis from this criterion (Fig. 3). Fig. 4 summarises the statistical analysis of the data obtained in groups 2 and 3. No significant difference was observed when comparing mean Amax values in the three subgroups of pulmonary stenosis. After surgical correction of the pulmonary stenosis, mean Amax was not significantly different from preoperative values. No significant difference was observed in comparing

mean Amax values of patients with mild, moderate, and severe pulmonary stenosis with that obtained in patients with secundum atrial septal defect. As in the other groups, correlation was poor between Amax and the patient's age ( $r = 0.16$ ) and weight ( $r = -0.07$ ).

#### "Ao max"

Maximal posterior displacement of the posterior aortic wall after atrial systole ranged from 0 to 4 mm (mean 2.3 mm) in the three groups of patients.

#### Discussion

The echocardiographic features of the normal pulmonary valve were first reported by Gramiak *et al.*<sup>4</sup> Characteristic patterns of stenotic pulmonary valve echo motion as well as those from normal subjects and from patients with left-to-right atrial shunts have been reported by Weyman and colleagues.<sup>2</sup> Their findings showed an "a" wave depth range of 0 to 7 mm (mean 3 mm) in normal subjects as well as in patients with mild pulmonary stenosis and in patients with atrial septal defect. In patients with moderate and severe pulmonary stenosis, the maximal

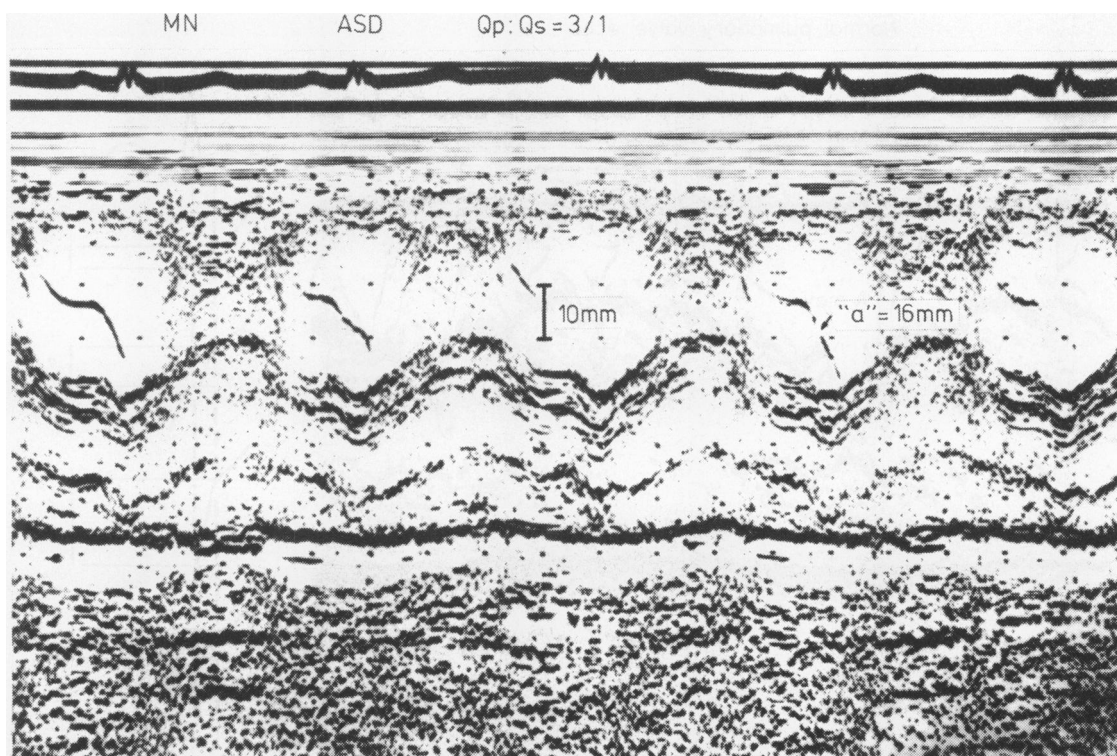


Fig. 2 Pulmonary valve echo recording obtained from a 12-year-old girl with a secundum atrial whose  $Qp/Qs$  was 3:1. Complete presystolic opening of the valve was observed in this patient with an  $A_{max}$  of 16 mm.

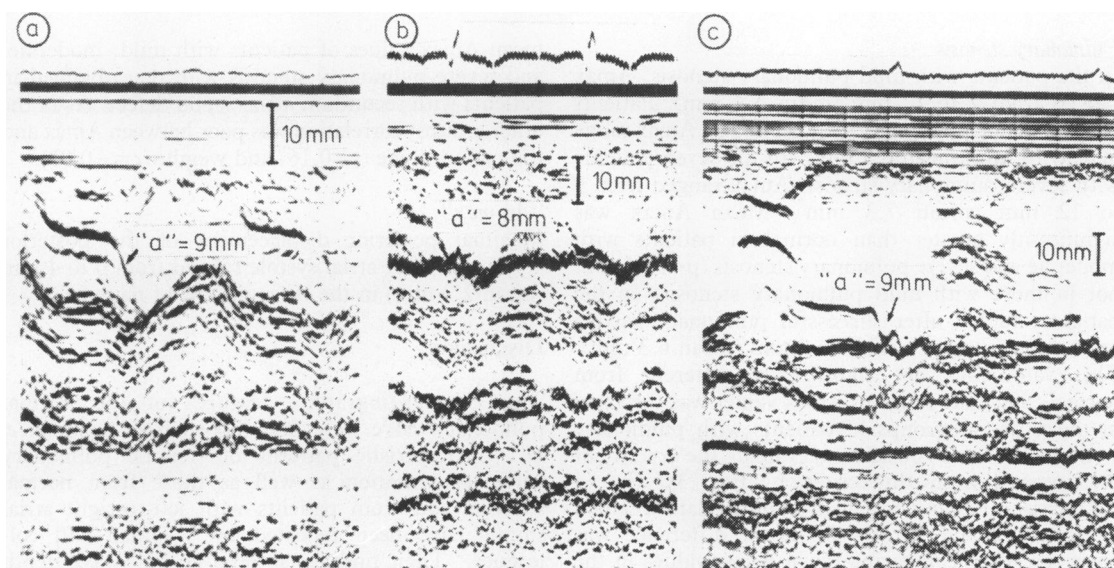


Fig. 3 Echocardiographic recording of the pulmonary valve in three patients with various degrees of pulmonary stenosis. The tracing on the left was obtained from a patient with mild pulmonary stenosis whose  $A_{max}$  was 9 mm. The tracing in the middle was obtained from a patient with moderate pulmonary stenosis; the  $A_{max}$  here is 8 mm. The tracing on the right was obtained from a patient with severe pulmonary stenosis;  $A_{max}$  is 9 mm.

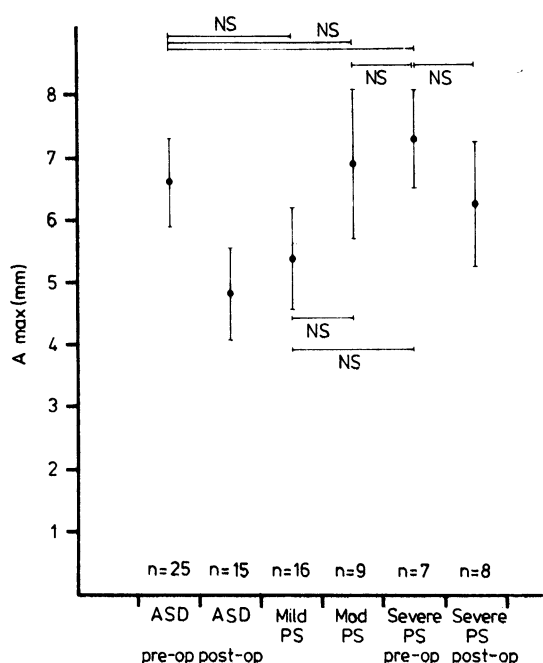


Fig. 4 Statistical analysis of the data obtained in groups 2 and 3. The dots represent mean Amax for each group and the vertical bars the standard error of the mean. NS, not significant; ASD, atrial septal defect; PS, pulmonary stenosis.

amplitude of the "a" wave increased considerably, ranging from 8 to 13 mm (mean 10 mm) suggesting that the severity of the stenosis could be predicted from the Amax. Furthermore in normal subjects, the leaflet always returned to the baseline or closed position before the onset of ventricular systole at some time during quiet respiration. These observations have not been confirmed by others.<sup>6,7</sup> Premature opening of the pulmonary valve has been described by Wann *et al.*<sup>8</sup> in a variety of anomalies such as constrictive pericarditis and tricuspid regurgitation. According to Gutgesell and Paquet,<sup>9</sup> increased Amax is not specific for severe valvular pulmonary stenosis and these authors pointed out that hypertrophy of the free wall of the right ventricle and of the septum is often present with this anomaly.

#### NORMAL SUBJECTS

Amax ranged from 0 to 12 mm in our normal subjects, giving a much wider range of normality compared with previous reported data. In our normal subjects, the pulmonary valve echo never returned to the baseline after atrial contraction during quiet res-

piration. Furthermore, complete presystolic opening of the valve was observed in 13 out of 57 normal subjects. These two observations might be explained by the fact that the PR interval is shorter in children than in adults. It has been suggested that the pulmonary "a" wave results from the presence of a pressure gradient at end-diastole between the right ventricle and the main pulmonary artery. Our observations suggest that such a gradient is of a greater magnitude in normal children than in adults, and that it is independent of the subject's age or weight.

#### ATRIAL SEPTAL DEFECT

Contrary to previous findings, our patients with secundum atrial septal defect also had mean Amax values higher than normal subjects and not different from those of patients with pulmonary stenosis. Since the pulmonary "a" wave results from the presence of an end-diastolic gradient between the right ventricle and the main pulmonary artery, our findings of increased Amax values in patients with secundum atrial septal defect suggest a decreased right ventricular compliance in those subjects. The lack of correlation between the Amax and the Qp/Qs in these patients suggests that the response to a given volume load is variable and is influenced by the contractile state of the myocardium.

#### PULMONARY STENOSIS

In patients with mild pulmonary stenosis, mean Amax was not different from that of normal subjects. In patients with moderate and severe pulmonary stenosis, mean Amax was significantly larger than in normals, but there was much overlap between the two groups so that individual cases could not be identified correctly from this measurement. The range of Amax was noted to be similar in normal subjects, in patients with secundum atrial septal defect, and in those with valvular pulmonary stenosis. After successful pulmonary valvotomy, mean Amax remained higher than normal, and the result of surgery could not be predicted from the echocardiogram.

Pocoski and Shah<sup>5</sup> have shown that the "a" dip on the pulmonary valve and depth of the "a" wave on the posterior aortic wall were significantly correlated. These authors proposed a dual mechanism for the production of the pulmonary "a" wave: the influence of left atrial volume changes in late diastole and the presence of a pressure gradient between the right ventricle and the main pulmonary artery. Our findings did not indicate such a correlation between the movement of the posterior aortic wall and the maximal amplitude of the pulmonary "a" wave, suggesting that the second mechanism is more important in the production of Amax in children.

## CLINICAL IMPLICATIONS

These findings indicate that mean Amax is neither specific nor sensitive for the presence or severity of valvular pulmonary stenosis in children. In patients with echocardiographic signs of right ventricular volume overload, presystolic opening of the pulmonary valve does not suggest the presence of an associated valvular pulmonary stenosis. In patients who have undergone pulmonary valvotomy, Amax is a poor indicator of the surgical results.

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